Medical Progress

Diseases of the Esophagus

Present Concepts

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In the past two decades, much information has been accumulated about the physiologic, pathologic and anatomic manifestations of various esophageal diseases. The radiologist plays an important role in evaluating these disorders and, in most instances, roentgenographic examination in conjunction with clinical history permits accurate diagnosis. In this report a review of normal esophageal anatomy and function is followed by an analysis of several selected disorders affecting the esophagus and their roentgenographic features.

Anatomy

The esophagus is a muscular tube, 20 to 24 cm in length, lined predominantly by stratified squamous epithelium.

The junction of the pharynx and esophagus is demarcated by the cricopharyngeal muscle which measures about 1 cm in vertical length. This muscle, at the same level as the cricoid cartilage, is the most proximal esophageal muscle component. When the pharynx and esophagus contain barium, this muscle produces a ring-like narrowing of the barium column that demarcates the pharyngo-esophageal junction. The remaining esophageal musculature begins at the lowermost border of the cricoid cartilage and interdigitates with the cricopharyngeus. Although individual variation is pronounced, striated muscle predominates the proximal one-third and smooth muscle the distal two-thirds of the esophagus.

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An upper esophageal sphincter 1 to 3 cm long exists at the proximal esophagus. This sphincter is composed of the cricopharyngeus muscle and the highest circular muscles of the esophagus.

Because many names have been used to describe the various anatomic landmarks of the lower esophagus, much confusion exists about the classification and nomenclature of structures in this area. Terms used in this discussion to denote these structures, as well as synonyms frequently used, are listed in Figure 1.

A sphincteric segment 1 to 4 cm long exists at the distal esophagus. It joins the stomach at the cardia. This sphincter is located partially in the thorax, partially in the diaphragmatic esophageal hiatus and partially in the abdomen. It is joined to the diaphragm by a tough, fibroelastic membrane, the phreno-esophageal membrane. The term *vestibule,* meaning an entrance hall (to the stomach), has been used to describe this region. Much confusion persists about this sphincter because it has many synonyms (Figure 1). We prefer to characterize this area as either the vestibule or lower esophageal sphincter.

An additional short contractile area, the inferior esophageal sphincter, makes up the proximal-most margin of the vestibule. Considerable confusion also exists about this nomenclature, for some authors refer to the entire vestibule or lower esophageal sphincter as the inferior esophageal sphincter (Figures 1 and 2).

Mucosa in the closed vestibule is thrown into longitudinal folds. Barium caught between these folds produces 2 to 4 smooth, vertical, linear

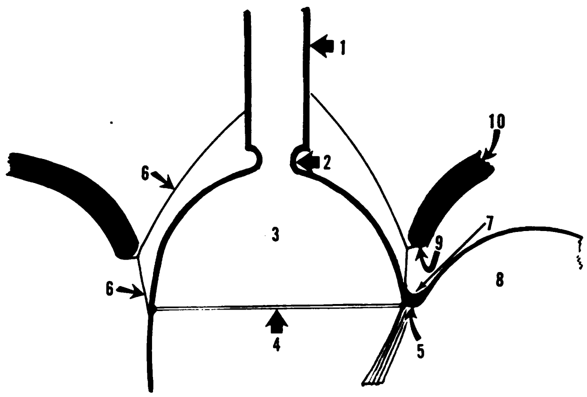

Figure 1.—Simplified diagrammatic representation of lower esophageal anatomy. Terms used in this report are in italics below. Synonyms frequently used for each area follow, with the name of the author (if known) responsible for introducing or popularizing each term.

1. *Lower Esophagus*
   supra ampullary esophagus (Botha, 1962)
   tubular esophagus (Wolf, 1967)

2. *Inferior Esophageal Sphincter* (Lerche, 1950)
   constriction caused by hiatus (Luschka, 1857)
   narrowing of Laimer (1883)
   sphincter-like inferior esophageal constriction (Strecker, 1905)
   constrictor cardiae (Gould and Barnhard, 1957)
   sub-ampullary constriction of Hacker (Turano, 1959)
   Ring A (Wolf, 1967)
   tubulo-vestibular sphincter (Wolf, 1967)

3. *Vestibule* (Lerche, 1950) *or Lower Esophageal Sphincter*
   cardia (Sommering, 1796)
   cardiac antrum (Arnold, 1838)
   esophageal ampulla (Barclay, 1915)
   epicardia (Ackerlund, 1929)
   cardiac sphincter (Abel, 1929)
   phrenic ampulla (Templeton, 1944)
   the term "phrenic ampulla" was first suggested by Waterson (1905) to describe hiatal hernia and has been used indiscriminately by radiologists for many years
   epiphrenic ampulla (Hillemand, Beau and Bernard, 1953) inferior esophageal sphincter

4. *Transverse Mucosal Fold* (TMF)
   lower esophageal ring (Schatzki, 1953)
   Schatzki ring
   lower esophageal web
   lower esophageal diaphragm
   Ring B (Wolf, 1967)
   cardia (originally used by Thucydides—423 B.C.—and Hippocrates—430 B.C.—to denote cardiac end of the stomach)

5. *Sling Fibers of Stomach*
   A thick muscle band lying within the other gastric muscle layers which also marks the esophago-gastric junction at its left lateral margin. A smaller muscle band, the constrictor cardiae, arises above it and encircles the esophago-gastric junction.
   sling fibers of Willis (Willis, 1674)
   muscle of Verheyen (1699)
   oblique fibers of stomach (Helvetius, 1719)
   collar of Helvetius
   Swiss Cravat
   bundle of His (His, 1903)

6. *Phreno-Esophageal Membrane*
   ligaments of Galen (Galen, A.D. 200)
   hiatal aponeurosis (Blandin, 1826)
   diaphragmatico-esophageal elastic membrane (Treitz, 1853)
   Laimer's membrane (Laimer, 1883)
   phrenico-esophageal diaphragm (Jonnesco, 1895)
   phreno-esophageal fascia (Le Double, 1897)
   phreno-esophageal fascial tube (Favera, 1906)

7. *Cardiac Notch*
   incisura cardiae (His, 1903)

8. *Fundus of Stomach*
   apex of stomach

9. *Margin of Esophageal Hiatus in Diaphragm*

10. *Diaphragm*

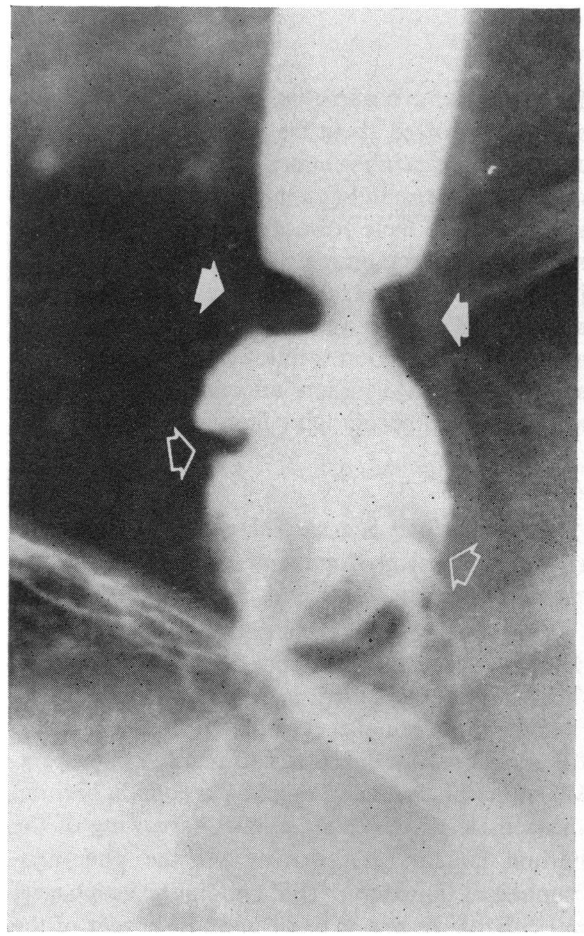

Figure 2.—Sliding hiatal hernia. The entire vestibule is intrathoracic and lies above a pouch of herniated stomach. The upper and lower vestibular boundaries, the inferior esophageal sphincter (solid arrows), and transverse mucosal fold (open arrows) respectively, are visualized. Thick mucosal folds are present in the herniated gastric pouch. (Through courtesy of McGraw-Hill publishers, "Diagnostic Radiology—A Companion to Harrison's Principle of Internal Medicine," P. Ruben Koehler, M.D., editor.)

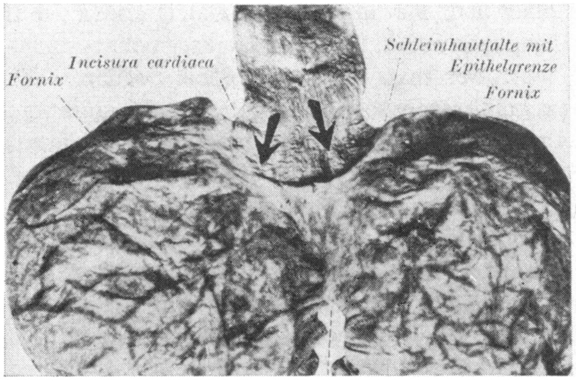

Figure 3.—Transverse mucosal fold. Specimen of normal esophagus and stomach which has been distended and fixed. A transverse mucosal fold is present at the esophago-gastric junction. (Through courtesy of Dr. Heinrich Hayek and the publishers, Springer-Verlag, Berlin, from—Zeitschrift fur Anatomie und Entwicklungsgeschichte 100: 218-255, October 1933.)

stripes on a radiograph.[17] As the vestibule distends, the folds are effaced. When a hiatal hernia is present the vestibule is completely displaced into the thorax and the gastroesophageal junction is demarcated by a transverse mucosal fold which forms at the distal vestibule[5,17,23] (Figures 1, 2 and 3). This fold, which is seen as a thin ledge-like transverse ring, is not identifiable on a roentgenogram of a normal barium-filled esophagus and stomach.

The esophageal body, lying between the two sphincteric segments, may be conveniently divided into proximal, middle and distal thirds. The junction of the proximal and middle thirds is near the aortic arch as viewed roentgenographically.

Normal Esophageal Physiology

The upper esophageal sphincter maintains a relatively high resting pressure (10 to 30 mm of mercury) when closed as compared with the adjacent intrapharyngeal and intraesophageal pressure.

The vestibule also has a higher resting pressure (15 to 35 mm of mercury) than the adjacent intraesophageal and intragastric pressures.

The highly integrated mechanism of deglutition transfers material from the mouth into the pharynx and then through the pharynx and esophagus to enter the stomach. The autonomic nervous system coordinates the entire sequence of neuromotor events.

Deglutition initiates a wave of inhibition, controlled centrally by a medullary swallowing center and mediated via the glossopharyngeal and vagus nerves, that progresses aborally through the pharynx and esophagus at a speed of 10 to 20 cm per second.[26] Consequent to the wave of inhibition, the high resting pressure of the closed upper esophageal sphincter drops abruptly (relaxes) within 0.2 to 0.3 seconds after swallowing. Following upper sphincter relaxation, the wave of inhibition sweeps down the esophageal body to reach the closed vestibule 1.5 to 2.5 seconds after deglutition. The vestibule then also relaxes.

Following the wave of inhibition, a peristaltic wave traverses the pharynx and esophagus. This wave reaches the relaxed upper esophageal sphincter about 1 to 1.5 seconds after swallowing. The sphincter contracts, in peristaltic sequence with the pharynx above and the esophagus below, as the peristaltic wave passes through it. The wave sweeps down the esophagus at a rate of 2 to 4 cm per second with a mean amplitude usually ranging from 20 to 50 mm of mercury. When peristalsis reaches the relaxed vestibule, this sphincter also contracts.

As the sphincters contract, they close with return of their high resting pressures which reestablish them as protective barriers at both ends of the esophagus. Closed sphincters maintain their physiologic, high pressure barrier by resisting distension[22] thereby preventing inadvertent collections of saliva and food in the esophagus and gastroesophageal reflux. Sphincter relaxation during deglutition, however, allows a bolus to enter the esophagus and stomach without resistance.

Esophageal peristalsis may be defined as a lumen-obliterating contraction, about 4 to 8 cm in length, moving at 2 to 4 cm per second.[26] Two types of esophageal peristalsis occur. Primary peristalsis is initiated by deglutition and secondary peristalsis occurs in response to local esophageal stimulation. The most common stimulus initiating secondary peristalsis is esophageal intraluminal distension. Material left behind in the esophagus following a primary peristaltic wave or material from gastroesophageal reflux is transported to the stomach by secondary peristalsis. Once elicited, both types of peristalsis appear similar and travel down the esophagus whether or not a bolus is being transported.

Esophageal response to swallowing or distension is variable in normal adults. Although swallowing and esophageal distension usually elicit peristalsis, the response is inconsistent with respect to incidence, force or extent of propagation. The in-

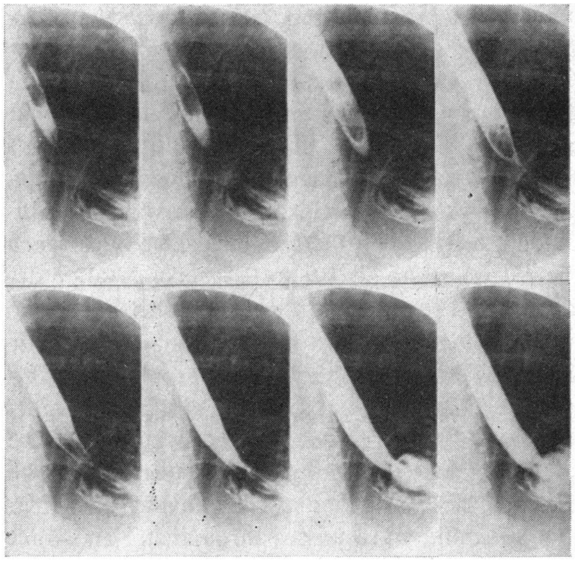

Figure 4.—Roentgenographic appearance of vestibular relaxation. With patient supine, serial 70 mm spot films were taken of the lower esophagus during a two-second interval immediately after deglutition of barium. The head of the barium column reaches the vestibule before its relaxation and assumes a V configuration (frames 1 and 2). The empty segment between the V and the stomach represents the vestibule. As the vestibule relaxes, barium flows through its lumen to enter the stomach. (Through the courtesy of W. B. Saunders Company, publishers, Radiologic Clinics of North America 7:147-161, April 1969.)

cidence and extent of propagation are less in the aged than in young adults.

Esophageal motor activity may be evaluated roentgenographically, either during fluoroscopy or on cineradiographic examination. The patient should be in the prone right anterior oblique position for evaluating esophageal motility. Following deglutition of barium, the contrast medium is distributed in a relatively continuous column throughout the esophagus. Because the upper esophageal sphincter relaxes promptly after swallowing, barium meets little resistance as it is propelled into the esophagus by buccopharyngeal dynamics. The barium column usually reaches the distal esophagus before vestibular relaxation occurs. In this circumstance, the barium encounters momentary delay before entering the stomach, and the head of the barium column adjacent to the closed vestibule assumes a "V" configuration (Figure 4). The point of the "V" demarcates the proximal margin of the vestibule. As the sphincter relaxes, the barium flows through it into the stomach. The vestibule may also be identified roentgenographically when relaxed and fully distended with barium. The intrathoracic portion then has a greater caliber than the adjacent esophagus above; and, during inspiration, the intrahiatal segment is slightly narrower than the intrathoracic portion. The vestibular responses to deglutition and inspiration, as manifested during roentgenographic and intraluminal manometric examination, are summarized in Table 1.

When primary peristalsis occurs, it causes the upper end (tail) of the barium column to assume an inverted "V" configuration. The lumen obliterating peristaltic wave is then visualized as a progressive movement of the inverted V-shaped tail down the esophagus (Figure 5).

Peristalsis is not the only muscular contraction that occurs in the esophagus. A muscular contraction, whether annular or segmental, that occurs simultaneously during manometric examination is termed a nonperistaltic contraction. Roentgenographically, this contraction simultaneously displaces barium both orally and aborally from the contraction site. Muscular activity representing repetitive nonperistaltic contractions also occurs.

TABLE 1.—*Vestibular Responses to Deglutition and Inspiration*

Deglutition

Radiology:

(1) vestibule opens

(2) vestibule increases in length and width (lengthening of transverse and longitudinal muscles)

(3) vestibule rises until almost entire length lies in thorax

(4) when fully distended, intrathoracic portion of vestibule is wider than esophagus above

(5) longitudinal mucosal folds effaced as vestibule opens

Intraluminal Manometry:

(1) high resting pressure is ablated

Inspiration

Radiology:

Hiatus*

(1) slides down on esophagus

(2) moves forward

(3) narrows

(4) rotates to left

Vestibule

(1) vestibule stretches

(2) on full inspiration, most of vestibule lies in thorax

(3) intrahiatal portion of vestibule is slightly narrower than intrathoracic portion

Intraluminal Manometry:

(1) pressure in intra-abdominal portion of vestibule increases

(2) pressure in intrathoracic portion of vestibule decreases

*This has been demonstrated experimentally by marking the hiatus.

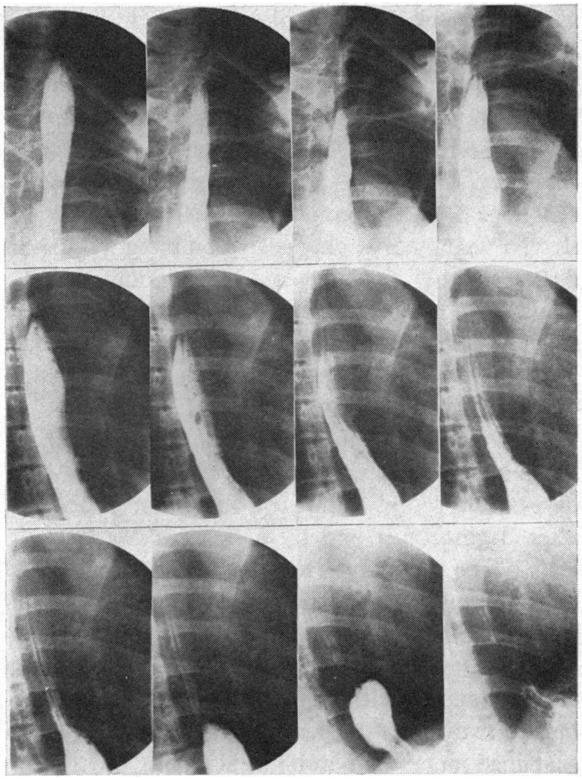

Figure 5.—Roentgenographic appearance of normal peristalsis. With patient supine, serial 70 mm spot films were taken during a 4.5 second interval following deglutition of barium. The x-ray tube was shifted slightly caudal after frames 3 and 10. Peristalsis causes the tail of the barium column to assume an inverted V shape. The peristaltic wave is visualized as a progressive aboral movement of the inverted V through the esophagus. (Through the courtesy of W. B. Saunders Company, publishers, Radiologic Clinics of North America 7:147-161, April, 1969.)

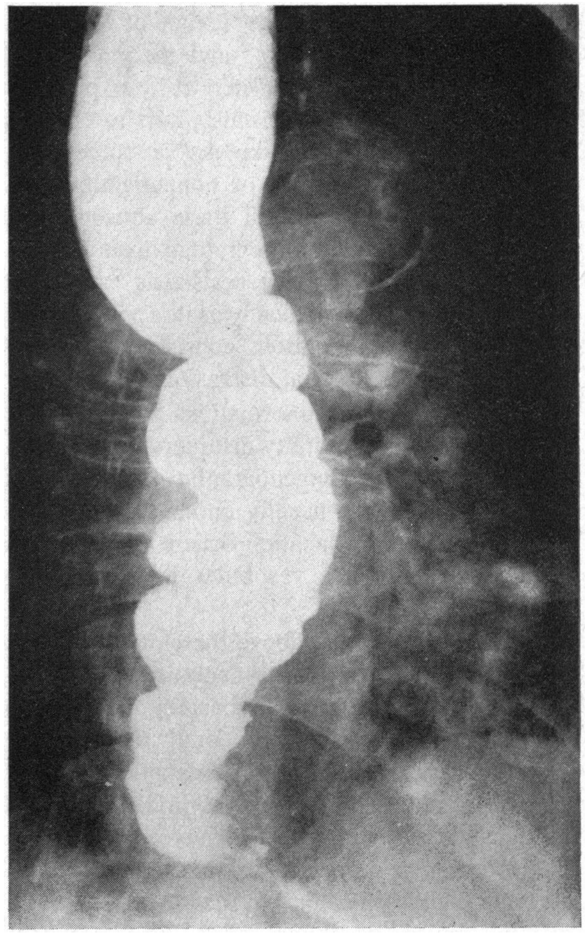

Figure 6.—Presbyesophagus. Tertiary contractions involve the lower one-half of the esophagus. Esophageal dilatation is prominent.

Their roentgenographic counterpart, termed tertiary contractions, cause the barium-filled esophagus to have an irregular, wrinkled contour. This appearance has also been called "curling" (Figure 6).

The cause of nonperistaltic contractions, single or repetitive, is not known. Their occasional occurrence in normal adults, however, denotes disorganization of the highly integrated mechanism of deglutition, albeit temporary.

In normal young adults 90 percent or more of swallows initiate primary peristalsis. Nonperistaltic contractions usually follow less than 10 percent of swallows, but emotional influences[32] and advancing age[40] may increase their incidence. Repetitive nonperistaltic contractions are not noted in young adults although they do occur in response to 10 percent or less of swallows in middle-aged adults.[14] Vestibular relaxation occurs after more than 95 percent of swallows in normal young adults. This incidence decreases slightly with aging.

Primary Esophageal Motility Disorders

A growing number of conditions resulting in abnormal neuromuscular esophageal function are now recognized. Such motility disorders may be classified as primary, the esophagus being the primary organ involved, or secondary to disorders which manifest associated esophageal abnormalities and to physical, chemical or pharmacologic effects on the esophagus.[43] In this review only the primary motility disorders (achalasia, diffuse esophageal spasm and presbyesophagus) will be discussed.

In esophageal motility disorders, the peristaltic mechanism and sphincter function are altered either singly or in combination.[43] Manometrically, abnormalities of primary peristalsis include complete inability or decreased incidence of peristalsis being initiated by swallowing and the inability of the peristaltic waves, once initiated, to progress to the stomach. When peristalsis fails to traverse the entire esophagus, but "breaks" at some level, either no muscular activity or nonperistaltic contractions occur distally. All these abnormalities are assessable by roentgenographic examination. The integrity of secondary peristalsis cannot be evaluated roentgenographically as it is not possible, during routine examination, consistently to produce localized esophageal distension.

Significant clinical abnormalities of sphincteric function in primary motility disorders are confined to the vestibule. Roentgenographic assessment of vestibular function is usually limited to determining whether or not relaxation occurs and, if so, its incidence. Incomplete vestibular relaxation may be suggested.

If barium is retained above the closed vestibule longer than 2.5 seconds after deglutition, the vestibule has failed to relax. The barium column then remains above the vestibule until the pressure generated by an advancing peristaltic wave or nonperistaltic contraction above, and transmitted through the barium column, overcomes the resisting vestibular pressure. Barium then forces the vestibule slightly open and squirts into the stomach.

If incomplete vestibular relaxation occurs, the lumen does not open as widely as normal. It is often difficult, however, to determine whether a narrowed vestibule is caused by incomplete relaxation, failure of relaxation or stricture secondary to esophagitis.

In the absence of esophageal muscular contraction and vestibular relaxation, barium will traverse the vestibule only if the patient is placed erect allowing the hydrostatic pressure of the resultant vertical barium column to overcome vestibular resistance. The barium will then force the vestibule to distend slightly, allowing barium to enter the stomach.

Achalasia

Achalasia usually has an insidious clinical onset, and though occurring at any age, frequently develops between 30 and 50 years of age. No sex predilection exists. The main symptom is dysphagia which, early in the disease, is frequently intermittent but later becomes persistent. Regurgitation, particularly at night, occurs commonly. Pain is a less frequent symptom.

Although etiologically obscure, achalasia is generally thought to be caused by an esophageal cholinergic innervation defect. The primary defect has been considered to be an absence or decrease of ganglion cells of Auerbach's myenteric plexus. However, this finding is not consistent and recent esophageal ultrastructure studies have demonstrated vagus nerve changes resembling Wallerian degeneration[10] and decreased cell counts of the medullary dorsal motor nucleus.[8] These features suggest that the primary defect is in the peripherial vagus nerve or dorsal motor nucleus and that esophageal changes are secondary.[8]

Utilizing intraluminal manometric recording techniques, achalasia is characterized by absence of peristalsis and failure of vestibular relaxation after deglutition[21] and by a positive Mecholyl®* test.[29] The Mecholyl® test is generally considered the confirming diagnostic test of classic achalasia. Following subcutaneous administration of Mecholyl®, 10 mgm or less, a positive test produces a tetanic nonperistaltic contraction of the distal one-half to two-thirds of the esophagus which causes a sustained increase in esophageal resting pressure of more than 10 mm of mercury. This response has been interpreted as demonstrating loss of esophageal cholinergic innervation. Positive Mecholyl® tests have also been recorded in diffuse esophageal spasm.[27]

Roentgenographically, achalasia is characterized by a persistent failure of the vestibule to relax and absence of esophageal peristalsis after swallowing contrast material. The upper esophageal sphincter, however, does relax after deglutition. Peristalsis, even occurring over a few centimeters of the upper esophagus, negates the diagnosis of classic achalasia.[43] Nonperistaltic or repetitive nonperistaltic (tertiary) contractions may occur in response to deglutition or spontaneously. The esophagus may be atonic in advanced cases. Esophageal dilatation of varying degree occurs secondary to the motor abnormalities.

Because the vestibule consistently fails to relax, the head of the barium column adjacent to the vestibule maintains a "V" configuration. Barium remains above the vestibule until pressure transmitted through the contrast column by nonperistaltic contractions in the esophagus above (or hydrostatic pressure if the patient is erect) overcomes the resisting pressure of the unrelaxed vestibule. The vestibule is then forced slightly open by the

*Mecholyl® = methylcholine chloride

Figure 7.—Achalasia. As barium squirts through the unrelaxed vestibule, the V shape of the head of the barium column is elongated and assumed a bird-beak configuration. This appearance is not specific for achalasia.

barium and barium squirts into the stomach. When barium is forced into or through the sphincter in this manner, the head of the barium column retains a V shape but the point becomes elongated (Figure 7).

This roentgenographic appearance, likened to a bird beak, has been erroneously considered characteristic of achalasia. Any disorder wherein the vestibule consistently fails to relax after deglutition manifests such beaklike deformity roentgenographically.[43] Although no disease except achalasia is uniformly characterized by absence of vestibular relaxation, an occasional patient with presbyesophagus,[40] diffuse esophageal spasm[14] or connective tissue disorder[13] has had failure of vestibular relaxation recorded in response to all swallows during manometric examination. The vestibule may also have a bird-beak configuration when involved with an annular constricting carcinoma or stricture secondary to esophagitis. These entities may usually be differentiated from achalasia on the basis of the motility profile manifest in the esophageal body.

Recently, six patients were described who had esophageal motor disturbance not characteristic of recognized esophageal diseases.[24] In all of them the clinical manifestations were similar to those of achalasia. During intraluminal manometry, however, peristalsis or vestibular relaxation, or both, were demonstrated after some swallows although the incidence was significantly reduced. The observers did not attempt to classify these patients into a single category of esophageal disease, as they did not manifest identical abnormalities. The investigators stated that these patients may represent examples of an esophageal disease the full expression of which is true achalasia.

Clinically significant primary esophageal motility disorders usually are readily classified into one of the recognized categories of esophageal disease. Occasionally, motor abnormalities occur that resist strict definition; they may well represent variations of true achalasia.

Diffuse Esophageal Spasm

Diffuse esophageal spasm is characterized clinically by intermittent dysphagia or pain, or both. No sex predilection exists and patients usually are middle aged.

Pain frequently is recorded as moderate substernal discomfort but may be colicky or mimic angina.[16] Frequently, symptoms are caused or aggravated by eating but may occur spontaneously. Symptoms usually are not incapacitating but an occasional patient who frequently experiences severe symptoms when eating may lose weight because he fears eating.

Intraluminal manometry[18] demonstrates primary peristalsis to be consistently propagated through the upper one-third of the esophagus in about 25 percent of patients. In all other patients with this disorder, the incidence of peristalsis in this region is diminished; nonperistaltic or repetitive nonperistaltic contractions, frequently of prolonged duration and abnormally high amplitude, are the predominant motor response to swallowing. Nonperistaltic contractions, single or repetitive, invariably follow deglutition in the lower two-thirds to one-half of the esophagus. Rarely does peristalsis traverse the entire esophagus. Although the vestibule usually relaxes, consistent failure of relaxation has been recorded[14]

Frequently, the esophageal muscle is thickened, occasionally to 2 cm (normal, 2 to 3 mm), and thickening may extend from the distal esophagus to the aortic arch level or higher.[18] Histologically, smooth muscle appears relatively normal[12] and ganglion cells are present.[18]

Roentgenographically, peristalsis occurs in the upper esophagus in response to some, and occasionally to all, swallows. When peristalsis is elicit-

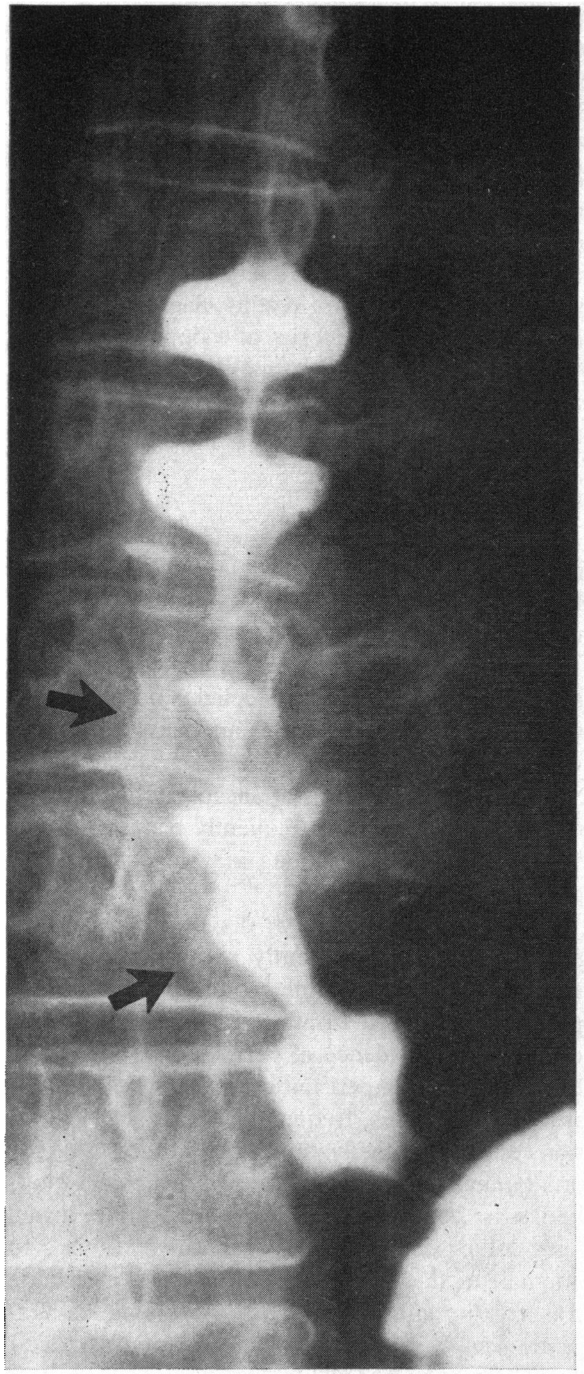

Figure 8.—Diffuse esophageal spasm. Tertiary contractions of abnormally high amplitude produce compartmentalization of the barium column. The esophageal wall is thickened (arrows). (Through the courtesy of W. B. Saunders Company, publishers, Radiologic Clinics of North America 7:147-161, April 1969.)

ed, its propagation invariably breaks about the level of the aortic arch and the lower esophagus responds with single or tertiary nonperistaltic contractions. If peristalsis does not occur in the upper esophagus in response to deglutition, nonperistaltic contractions are elicited throughout the entire esophagus.

The abnormally high amplitude tertiary contractions produce compartmentalization of the barium column (Figure 8). This appearance is produced by barium being displaced both proximally and distally from the sites of highest generated pressure to adjacent areas where pressures attained during contraction are lower. This roentgenographic feature has been called "tiered spasms," pseudodiverticulosis, and the "rosary bead" esophagus.

Thickening of the distal one-third to two-thirds of the esophageal wall is often demonstrated roentgenographically. The lumen of the involved part of the esophagus may be narrowed; in these instances, the lumen of the uninvolved upper esophagus may be mildly dilated.

The cause of diffuse esophageal spasm is not known. Kramer, et al[27] recently demonstrated that most patients with this disorder manifest a hypersensitive esophageal response to Mecholyl®. They suggest that achalasia and diffuse spasm are related disorders because of their similar response to this cholinergic agent. Possible transition of diffuse esophageal spasm to achalasia has also been recorded.[28] Esophageal electron microscopy in achalasia and diffuse spasm by Cassella and colleagues,[9] however, suggest that the two diseases are separate entities. These investigators propose that diffuse spasm is characterized by primary involvement of the vagal afferent (sensory) system.

Presbyesophagus

Presbyesophagus is an esophageal motor dysfunction associated with aging. This motility disorder is usually noted in persons of geriatric age but it may be present in late middle age. Although geriatric patients may manifest a normal esophageal profile to deglutition, most reveal some abnormality. Patients usually have no esophageal symptoms; an occasional patient may have dysphagia when eating solids.

The predominant manometric esophageal abnormality is an inability to initiate primary peristalsis and vestibular relaxation.[40] The incidence of both responses is usually decidedly diminished. Propagation of primary peristalsis is also impaired. The decreased incidence of peristalsis following deglutition is accompanied by an increase in nonperistaltic

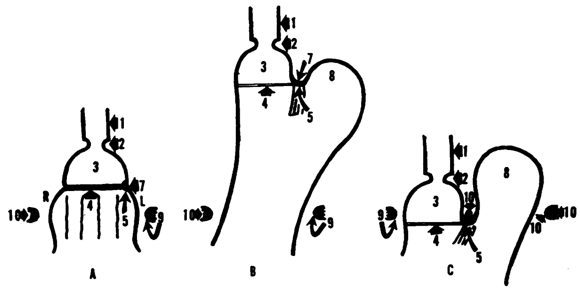

Figure 9.—Diagrammatic representation of the radiological anatomy of hiatal hernia.

A. *Small sliding hiatal hernia.* The vestibule (3) demarcated by the inferior esophageal sphincter (2) above, and the transverse mucosal fold (4) below, lies completely in the thorax above the hiatus (9). Below the vestibule is an intrathoracic sleeve of stomach, the walls of which are composed of a small portion of the lesser curve of the stomach on the right (R) and a small length of the fundus of the stomach on the left (L).

B. *Large sliding hiatal hernia.* The fundus of the stomach (8) lies in the thorax and balloons out to simulate its normal shape. The relative positions of the vestibule (3) and fundus (8) are similar to that seen when no hernia is present.

C. *Paraesophageal hiatal hernia.* The vestibule (3) maintains its normal relationship to the hiatus (9). The fundus of the stomach (8) rolls into the thorax either through the hiatus or through a separate opening in the diaphragm (10) immediately adjacent to the hiatus.

contractions, often repetitive, of normal amplitude. The Mecholyl® test is not positive.

A complete spectrum of peristaltic abnormalities is observed roentgenographically. Frequently peristaltic waves traverse only the upper esophagus, and occasionally no esophageal peristalsis is identified. The predominant roentgenographic feature is tertiary contractions, frequently involving a long esophageal segment[41] (Figure 6).

The vestibule may demonstrate a normal or decreased incidence of relaxation following deglutition or it may consistently fail to relax. Patients with significant motor dysfunction frequently have uniform esophageal dilatation which usually is moderate but may be prominent (Figure 6).

The roentgenographic features of presbyesophagus may occasionally resemble diffuse esophageal spasm, connective tissue disease, esophagitis and, rarely, achalasia.[42] Other diagnostic procedures may then be required before definitive diagnosis is possible.

Hiatal Hernia

Two types of hiatal hernia occur, sliding and paraesophageal. Hiatal hernias may occur in infancy but are most common in adult life; they increase in incidence with age.

Sliding hiatal hernia, also known as axial, concentric or short esophagus type hernia, is most common. In sliding hiatal hernia, the entire vestibule lies within the thorax above an intrathoracic sleeve of stomach which has also herniated from the abdomen. If the hernia is small, the intrathoracic gastric sleeve, composed of a portion of fundus, usually forms a tube or pouch directly below the vestibule (Figures 2 and 9 A). If the hernia is large, the gastric fundus slips entirely into the thorax and balloons out to the left of the vestibule where it assumes its normal shape (Figure 9 B).

True paraesophageal hiatal hernias are uncommon at any age. These are also referred to as parahiatal or rolling hiatal hernias. In these the vestibule maintains its normal anatomic relationship to the hiatus. The esophagogastric junction remains *below* the diaphragm, but part of the stomach herniates into the chest *alongside* the esophagus. The stomach may herniate either through the hiatus or through a separate diaphragmatic opening adjacent to the hiatus (Figure 9 C).

Sliding Hiatal Hernia

Radiologic diagnosis of sliding hiatal hernia has traditionally been divided into two parts: (1) demonstrating the abnormal radiologic anatomy and (2) demonstrating gastroesophageal reflux of barium.

Although a sliding hiatal hernia is occasionally seen when the patient swallows barium standing upright (irreducible or fixed hernia), it is best demonstrated by having the patient prone over a bolster placed below the twelfth rib. By increasing intra-abdominal pressure, this procedure forces the stomach into the thorax. It also ensures maximum distension of the distal esophagus after the patient swallows barium.

If a normal patient lies prone over a bolster and swallows a thick barium bolus, most of the vestibule rises into the thorax, but the distal-most portion remains intra-abdominal. Similarly, if a normal patient inspires maximally, the hiatus slides down the esophagus, but again the distal vestibule remains in the abdomen. During both maneuvers, the stomach does not herniate above the hiatus.[6]

If the hiatus were demonstrable on radiographs, little problem in diagnosing hiatal hernia would occur. Unfortunately, the hiatus is not observed roentgenographically. The position of the hiatus may be inferred when a hernia is present because

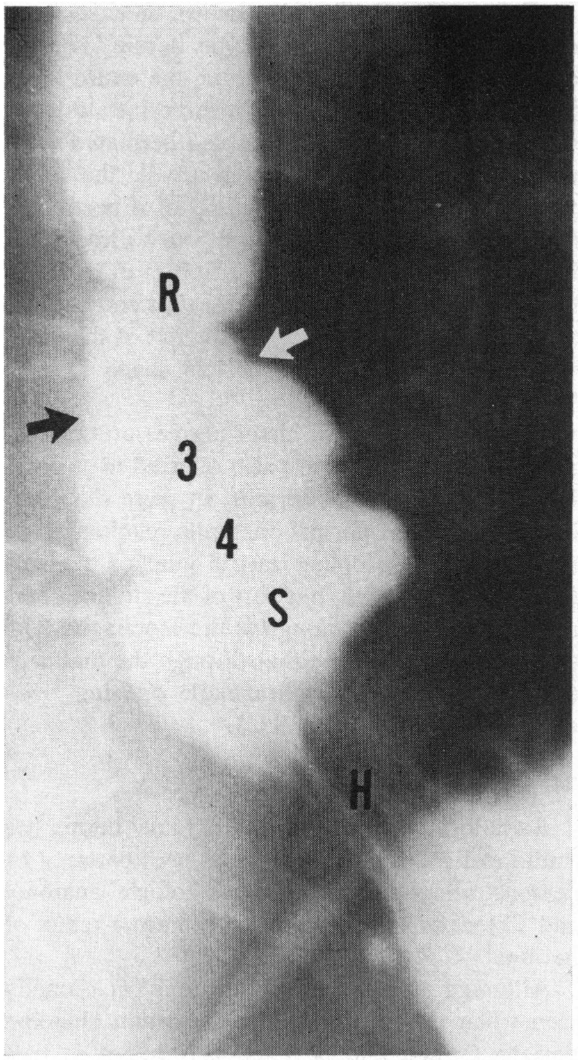

Figure 10.—Esophageal ring (stricture) located above the vestibule. Rigid, thin circumferential ring (R) is present above vestibule (3). Arrows indicate position of inferior esophageal sphincter. A hiatal hernia exists because vestibule (3), transverse mucosal fold (4) and portion of stomach (5) lie in thorax above hiatus (H). The intrahiatal stomach is so compressed by the hiatal margins that the gastric mucosal folds are squeezed together.

the hiatal margins may compress the intrahiatal stomach (Figure 10), but this feature is neither consistently present nor sufficiently reliable to be of diagnostic value.

In a small sliding hernia, the abnormally located gastric sleeve has a different configuration than normal stomach. The herniated stomach assumes a tubular or fusiform shape. Because a small hernia and the normal vestibule often have a similar configuration, it usually is impossible to distinguish them accurately by shape alone. Since it is difficult to identify the position of the hiatus and to recognize a small intrathoracic gastric sleeve by its shape alone on roentgenographic examination, other criteria must be used to diagnose a small sliding hiatal hernia.

The presence of a small sliding hiatal hernia may be accurately diagnosed if the transverse mucosal fold is demonstrated.[5,6,17] This fold, a thin, ledgelike ring, forms the distal vestibular boundary, thereby demarcating the gastroesophageal junction (Figures 2, 3 and 10). When the gastroesophageal junction is located intra-abdominally, the transverse mucosal fold is not roentgenographically demonstrable; however, it does produce a roentgenographically identifiable structure when it is intrathoracic. Thus, the fold can be seen in all patients with sliding hiatal hernia. The fold is best seen when the vestibule is maximally distended with barium (Figures 2 and 10); it disappears as the vestibular lumen closes. To aid accurate identification of this anatomic landmark it is helpful if the upper vestibular boundary—that is, the inferior esophageal sphincter—also be demonstrated, as it may resemble the transverse mucosal fold. This sphincter also produces a ringlike deformity of the barium column (Figures 2 and 10) but does not indicate the presence of hiatal hernia because this portion of the vestibule is normally located in the thorax. This sphincter or ring can be demonstrated in most patients whether or not a hiatal hernia is present. The ring is best identified when the patient swallows barium while lying prone over a bolster or when the barium's consistency is similar to that of masticated solid food. The ring has rounded margins and is most prominent when the esophageal lumen at this site is only partially distended. When the sphincteric lumen is almost completely open, the ring is visualized as a slight transverse luminal indentation which may be difficult to distinguish from the transverse mucosal fold. The indentation disappears when the vestibule (sphincter) is completely open.

The above described landmarks may be difficult to record roentgenographically, for they appear but fleetingly during the passage of barium through the lower esophagus and vestibule, but an experienced fluoroscopist can usually identify them. Continual recording by cinefluorography permits the entire sequence of the esophageal study to be replayed and reviewed at a later time, thus aiding identification of anatomic details.

The mucosal pattern may also aid in differentiating the normal vestibule from herniated stomach.

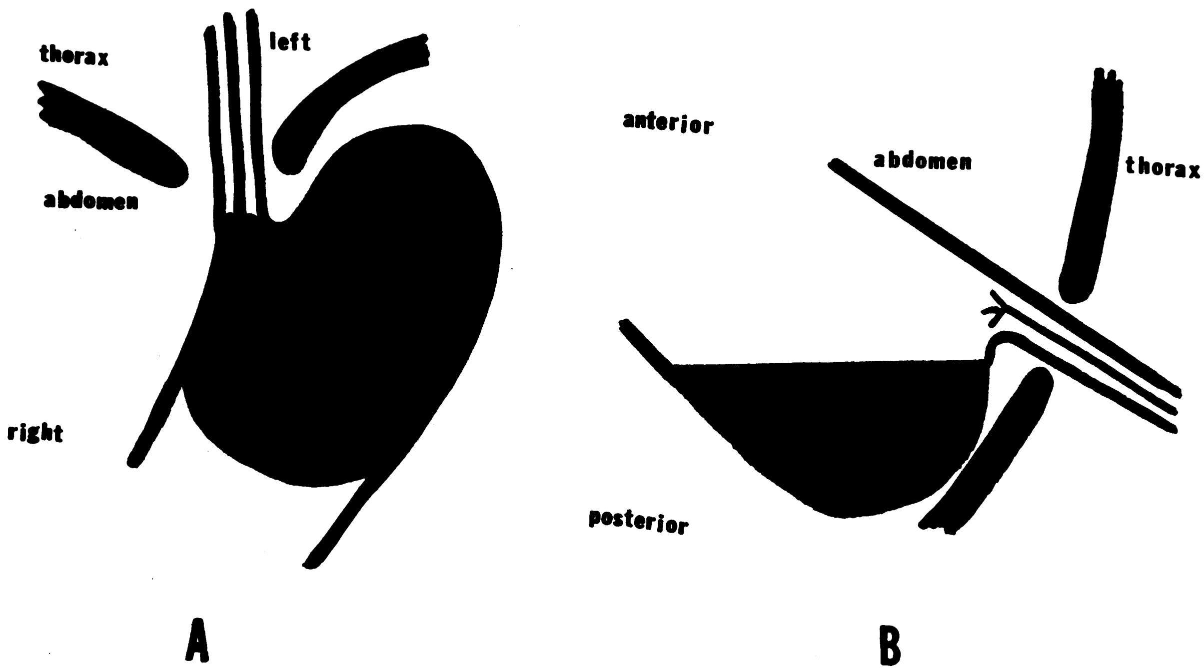

Figure 11.—Relationship of barium pooled in the gastric fundus to the esophago-gastric junction.

A. Appearance on posteroanterior or anteroposterior roentgenogram falsely suggests that barium in the stomach is covering the esophago-gastric junction.

B. True situation is demonstrated by horizontal x-ray with patient in same position. Barium pool is too shallow and does not cover the esophago-gastric junction.

The normal closed vestibule presents two to four fine, vertical, roughly parallel folds, whereas a herniated tube of stomach often has numerous thick, coarse mucosal folds (Figure 2). This distinction frequently is not possible, however, because gastric mucosa in a hernia may be stretched and thinned.

The radiologic diagnosis of a large sliding hiatal hernia usually presents no difficulty. It may be discovered on a routine chest roentgenogram. Large sliding hernias may be surprisingly asymptomatic. Very large hiatal hernias (totally intrathoracic stomach) may present as an acute emergency with gastric volvulus.

When either a small or large sliding hiatal hernia is present, the cardinal functional abnormality that may occur is gastroesophageal reflux. Many radiologists and clinicians feel it obligatory to demonstrate gastroesophageal reflux to prove the presence of a hiatal hernia. To demonstrate reflux, the patient frequently is subjected to a wide variety of gymnastic maneuvers. Examples: With the patient prone or supine the radiographic table is tilted head down; or the patient is called upon for straight leg raising while supine, or for crouching in the genupectoral position, or to touch his toes while erect; or pressure may be applied to the abdomen of the supine patient. Unfortunately there are several pitfalls in the interpretation of these tests. First, to demonstrate esophageal reflux it is essential that barium in the gastric fundus cover the esophago-gastric junction. The various positions described above were designed to produce this distribution of barium, but, unfortunately, neither anteroposterior nor posteroanterior roentgenograms provide certainty that this has been achieved. Whatever the patient's posture, only a horizontal x-ray beam will demonstrate whether or not the barium is covering the esophagogastric junction (Figure 11). Second, the hiatus may so compress the herniated sleeve of stomach passing through it that the gastric mucosal folds are squeezed together (Figure 10). These folds (the gastric mucosal choke) may then prevent reflux during the roentgenographic examination.

Further, standard barium preparations, unlike gastric juice or food, have a high specific gravity (6 or 7) and therefore reflux of barium may not occur during examination even though the hernia may allow reflux of gastric juice or food at other times. Recently Sandmark[36] demonstrated that standard barium may not reflux when a hernia is present whereas reflux may occur when a barium preparation of low specific gravity is used.

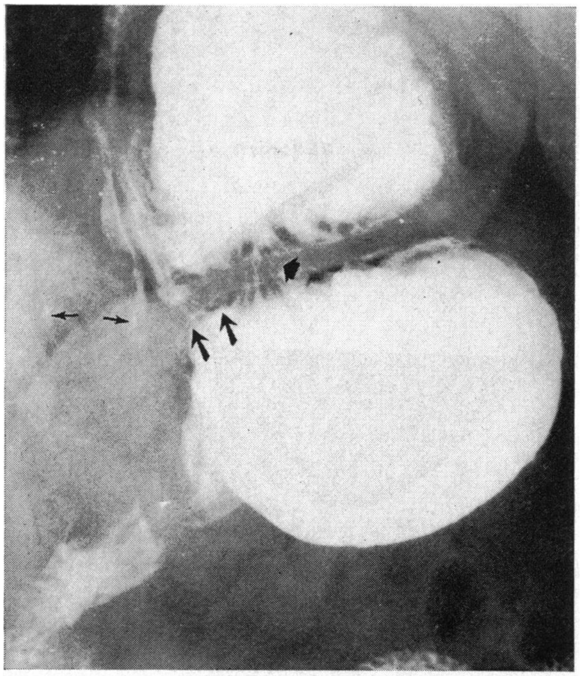

Figure 12.—Paraesophageal hiatal hernia. The esophagogastric junction (2 large arrows) lies in its normal anatomical position. Most of the gastric fundus has herniated into the thorax. The gastric mucosal folds are squeezed together, thinned and straightened by the diaphragm (single arrow). The aorta is calcified (2 small arrows) and lies behind the esophago-gastric junction but overlaps the lower thoracic esophagus.

Some radiologists test for reflux from the barium-filled stomach by having a recumbent patient drink water. Because deglutition causes vestibular relaxation, this technique may allow transient esophageal reflux to occur even when no hernia exists. If such reflux is considered to indicate the presence of a hiatal hernia (usually it is so interpreted), the technique then causes false positive results.

Because of these various factors, demonstration of gastroesophageal reflux is not necessary to prove a hernia present. Moreover, gastroesophageal reflux is dependent on so many variables that it is an unreliable test as usually performed. Both false positive and false negative results are possible under certain conditions.

Paraesophageal Hernias:

In paraesophageal hernias the esophagogastric junction remains below the hiatus and the vestibule is normally located, but a pouch of stomach extends alongside the esophagus into the thorax (Figure 12). Gastroesophageal reflux does not occur and the major complications are related to vascular congestion and ulceration in the herniated gastric fundus. Sometimes the herniated fundus may not fill with barium; the diagnosis is then usually missed.

Esophagitis

Esophagitis is classified as acute and chronic. Acute esophagitis may be caused by gastroesophageal reflux of acid-peptic juice, infectious agents, radiation and caustics. Patients usually are not examined roentgenographically during the early or acute phase of esophagitis. If esophageal barium studies are done, abnormalities usually are not identified. However, if inflammatory reaction and edema are sufficient, a lack of complete distensibility of the involved portion of the esophagus may be evident.

Chronic esophagitis is usually produced by recurrent gastroesophageal reflux: sliding hiatal hernias are present in most persons with this disorder. Clinical symptoms include heartburn, retrosternal pain, regurgitation and occasionally dysphagia. Controversy exists as to whether heartburn is due to chemical irritation[4] or abnormal esophageal motility.[39]

Chronic esophagitis may be manifested histologically by mucosal edema and erosion or, if more severe, mucosal ulceration associated with inflammatory edema of the submucosa or submucosa and muscularis externa. The latter response may produce thickening and decreased distensibility of the esophageal wall with resultant luminal narrowing. The narrowing is caused by inflammatory edema which produces wall thickening and limits esophageal distensibility. Narrowing may also be caused by stricture.

Esophageal motor dysfunction may be associated with chronic esophagitis. Motility abnormalities that may occur include incompetency of the lower esophageal sphincter and an absence or decreased incidence of peristalsis, accompanied by an increased incidence of nonperistaltic contractions, in the diseased esophageal segment.[33]

Neither superficial epithelial erosions nor mucosal edema are identifiable on roentgenograms. The only morphologic abnormality roentgenographically detectable is the luminal narrowing associated with thickening and decreased distensibility of the esophageal wall. This is usually characterized by a tapered symmetric narrowing of the involved segment without sharp demarcation between the abnormal and normal esophagus (Figure 13). Al-

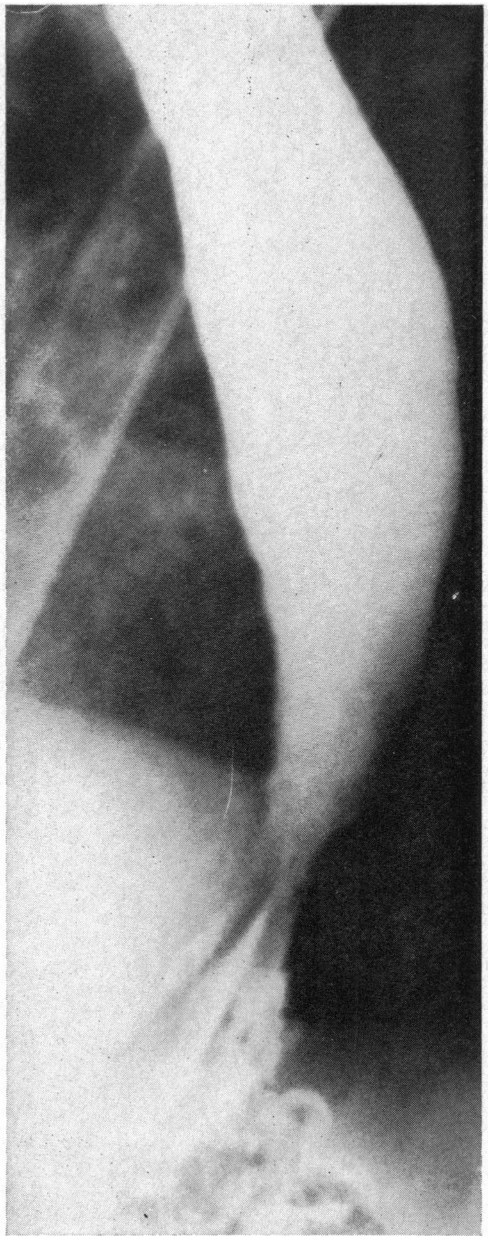

Figure 13.—Chronic esophagitis. Lower esophageal lumen is symmetrically narrowed. The demarcation between normal esophagus and the involved segment is not sharply delineated.

though the narrowed lumen may close fully, it does not open completely.

Peptic esophagitis may occasionally be manifested roentgenographically as a fixed ring deformity of the esophageal lumen. This feature will be discussed under "Esophageal Rings."

Roentgenographic evaluation of esophageal motility may be normal. Frequently, however, primary peristalsis fails to traverse the segment involved with esophagitis and nonperistaltic contractions (single or tertiary) then make up the predominant motor response of the affected region. Recently, acid barium (pH 1.7) has been proposed as a means of inducing abnormal roentgenographic motility patterns in patients with esophagitis who demonstrate normal motility when a standard barium preparation is swallowed.[15]

Esophageal Rings

An esophageal ring is a thin, circumferential indentation of the esophageal lumen. Descriptions of esophageal rings in the literature are very confusing but this is largely semantics. The main types of esophageal rings are classified in Table 2. Two rings are frequently observed in the lower esophagus:[5,6,17] an upper, the inferior esophageal sphincter, and a lower, the transverse mucosal fold (Figures 2 and 10).

Function of the inferior esophageal sphincter is unknown. Many radiologists regard it as a sphincter because it is capable of partial closure independent of the remaining vestibule. Anatomic studies suggest the ring is formed by esophageal muscle coat contraction.[17] When present it only partially occludes the lumen until peristalsis reaches it. It then closes completely as does the vestibule below. Most investigators are skeptical about considering this ring a sphincter because no high pressure zone is detected within it during intraluminal manometric investigation. It should be noted, however, that as the esophagus closes, its lumen, in cross-section, changes from a circular to a stellate configuration.[17] This permits a pronounced reduction of luminal flow to occur with only minimal change in pressure.[11] Thus, the inferior esophageal sphincter may have a sphincteric function when it produces a ringlike narrowing of the esophageal lumen. Inferior esophageal sphincter visualization does not indicate evidence of hiatal hernia or esophagitis. Transverse mucosal fold visualization indicates hiatal hernia; its presence does not imply esophagitis.

TABLE 2.—*Classification of Esophageal Rings*

1. Inferior esophageal sphincter
2. Transverse mucosal fold
3. Mucosal rings (diaphragms) secondary to localized peptic esophagitis
    (a) at or near the transverse mucosal fold
    (b) at or near the inferior esophageal sphincter
    (c) elsewhere
4. Muscular ring approximately related to mucosal junction in a lower esophagus abnormally lined with columnar epithelium
5. Congenital

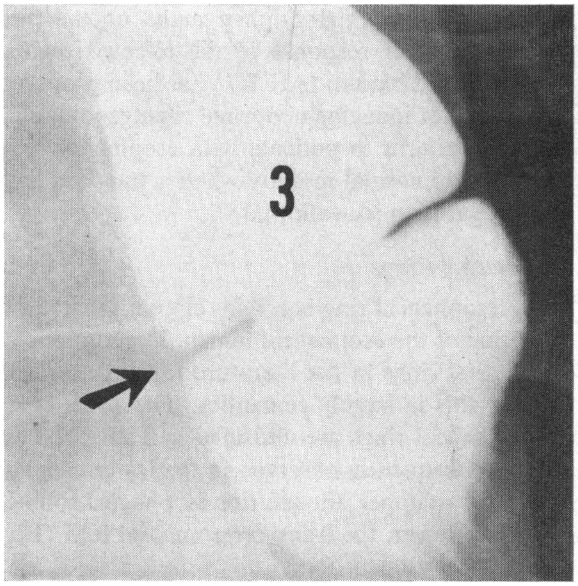

Figure 14.—Esophageal ring (stricture) at transverse mucosal fold. The vestibule (3) is fully distended and a hiatal hernia is present. Rigid ring (arrows) is located at the level of the transverse mucosal fold. It was demonstrated that the inferior esophageal sphincter was present and located proximal to the ring in other films. The luminal diameter of the esophagus at the site of ring formation is 1.0 cm.

Peptic esophagitis may cause thin, ledgelike mucosal diaphragms which protrude into the lumen and produce ring deformities. The rings are rigid, denoting localized stricture, and cause narrowing of the esophageal lumen (Figure 10). These mucosal diaphragms are usually single but, rarely, multiple mucosal diaphragms may develop in infants. The diaphragm usually develops at or near the transverse mucosal fold (Figure 14). When this occurs, the fold may not disappear as the vestibule closes and the lumen at this site may become very narrow. A barium-filled capsule or marshmallow may be unable to traverse the narrowed lumen. Clinically, the patient may complain of dysphagia. McMahon, Schatzki and Gary describe a patient who had dysphagia and a lower esophageal ring for nine years.[31] On histologic examination the ring, composed of mucosa, connective tissue and smooth muscle, was located at the gastroesophageal junction. Unfortunately, the term "Schatzki ring" is often used indiscriminately to describe any esophageal ring; because of this, the eponym should be abandoned.

Another common site for mucosal diaphragm formation is at or near the inferior esophageal sphincter but it may occur anywhere in the esophagus (Figure 10).

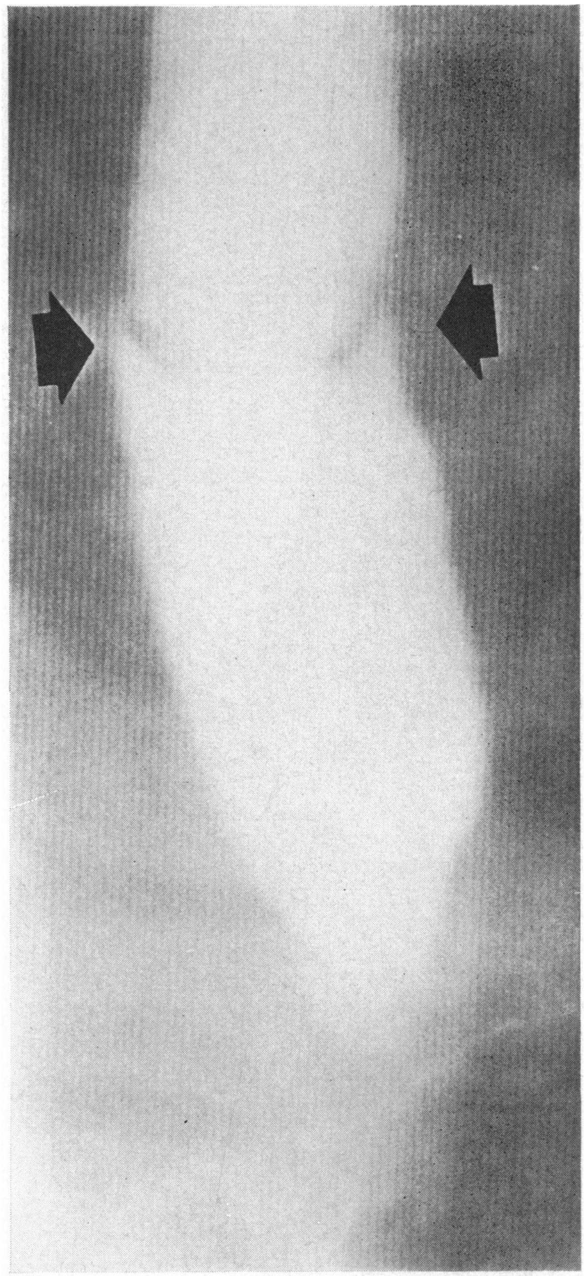

Figure 15.—Congenital esophageal ring. Esophagram requested in newborn period because the infant refused feedings. The thin, ledge-like diaphragm (arrows) is located in the lower esophagus.

Patients with an esophagus lined by columnar epithelium may develop esophageal stricture similar to that in patients with the more usual form of peptic esophagitis. The stricture is usually fibrous. However, the narrowing occasionally is not due to inflammation and fibrosis but is secondary to gross muscle thickening.[3] It then appears as a ring deformity.[38] Barrett describes this ring as

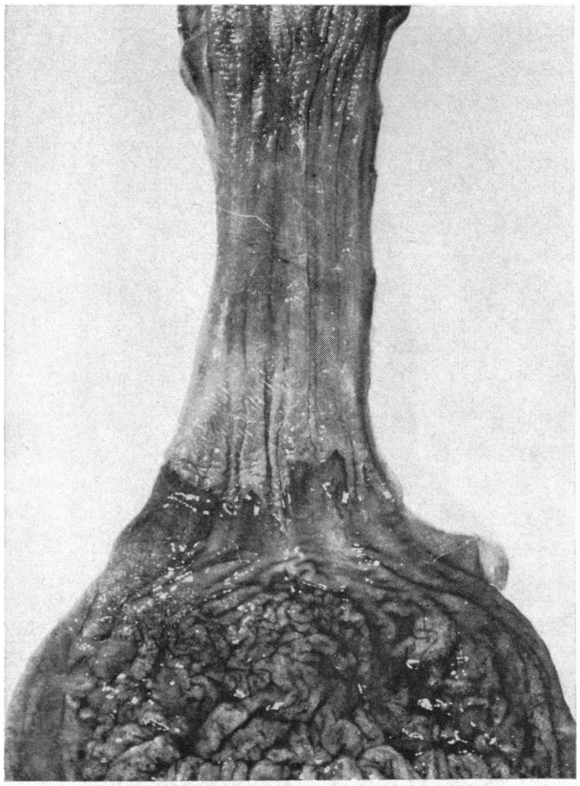

Figure 16.—Distal esophageal mucosa. The lower esophagus and upper stomach has been opened longitudinally. The junction between esophageal squamous and esophageal columnar epithelium is readily visible as an irregular "Z" line. The mucosal junction lies proximal to the esophagogastric junction. The longest digitation of squamous epithelium measures 1.5 cm. Longitudinal esophageal mucosal folds traverse both the squamous and columnar epithelial segments. In contrast to the longitudinal folds formed in the esophagus, gastric mucosa forms rugae. (Through courtesy of Norman R. Barrett, F.R.C.S., editor, Thorax 21:487-498, November 1966.)

having an appearance similar to the pylorus in congenital hypertrophic pyloric stenosis.[3] Although this ring may be situated anywhere in the esophagus, the involved area is frequently near the aortic arch region.

Congenital mucosal diaphragms are usually discovered in infancy and may occur anywhere in the esophagus (Figure 15). The cause is unknown.

Peptic Ulcer

The esophagus is lined primarily with stratified squamous epithelium and the stomach by columnar epithelium. The junction between these two types of mucosa does not correspond to the esophago-gastric junction. A variable length of the distal esophagus is lined by columnar epithelium without parietal cells (Figure 16). This segment, usually about 0.75 cm long, is rarely more than 2.5 cm.

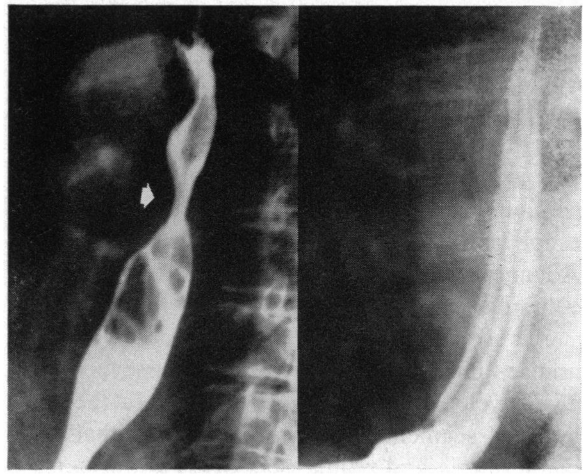

Figure 17.—Lower esophagus abnormally lined by columnar epithelium with esophageal stricture due to tryptic esophagitis. Total gastrectomy and esophagojejunostomy seven years previously for gastric carcinoma. Severe symptoms of gastroesophageal reflux since operation. Recent onset of dysphagia. *Left,* esophagram demonstrates esophageal stricture (arrow) just distal to aortic arch. Esophagoscopy and biopsy revealed that the esophagus below the stricture was lined by columnar epithelium with a few abnormal villi, resembling jejunal mucosa. *Right,* mucosal pattern of lower esophagus (distal to the stricture) appears normal roentgenographically. (Through courtesy of Dr. F. Dick Berridge, Director, Diagnostic Radiologic Departments, The United Cambridge Hospitals, England.)

Barrett in 1950 described a condition wherein a greater length of lower esophagus is lined by columnar epithelium. The patients, usually middle-aged or elderly, invariably have hiatal hernia. Barrett subsequently called the disorder "lower esophagus lined by columnar epithelium."[3] Although the cause is not firmly established, it probably represents an unusual sequela of reflux esophagitis whereby columnar epithelium replaces squamous epithelium during healing. Rarely, a similar phenomenon occurs following total gastrectomy secondary to tryptic (alkaline) esophagitis (Figure 17).

Deep, chronic peptic ulcers may occur in an esophagus lined abnormally by columnar epithelium. The penetrating ulcers are well defined and easily recognized roentgenographically (Figure 18). The ulcer may erode a large vessel in the esophageal wall and produce massive hemorrhage.[1,3] Further, the ulcer may perforate the mediastinum, pleura, heart, aorta or pulmonary artery, again with the possibility of associated massive hemorrhage. In contrast to the deep ulcers occurring in this disorder, the erosions or ulcerations of squamous epithelium which occur in peptic esophagitis are not identifiable on roentgenograms.

Esophageal strictures may also occur in an esophagus abnormally lined by columnar epithelium. They usually present above the mucosal junction and frequently are in the upper esophagus near the level of the aortic arch (Figure 17, left). The triad of an esophagus abnormally lined by columnar epithelium, chronic peptic ulcer and esophageal stricture is called Barrett's syndrome. Adenocarcinoma may, on occasion, develop in the columnar epithelium.

An esophagus with normal squamous and columnar epithelium distribution may also occasionally develop penetrating peptic ulcer in the short terminal segment lined by columnar epithelium if hiatal hernia and reflux esophagitis are present.

Carcinoma of the Esophagus

Esophageal carcinoma may be defined as to its site of origin. Thus, carcinoma may occur in the upper, middle or lower third of the esophagus. These areas correspond to or encompass the thoracic inlet, tracheal bifurcation and retrocardiac regions, respectively. However, carcinoma may develop anywhere in the esophagus, including the post-cricoid region, vestibule and esophago-gastric junction. The area near the tracheal bifurcation is most frequently involved. Esophageal carcinoma develops most often in elderly men. Post-cricoid lesions, however, occur mostly in women. An increased incidence of esophageal carcinoma is noted in persons with achalasia and post-cricoid webs associated with iron deficiency anemia (Plummer-Vinson syndrome).

Esophageal carcinomas are primarily of squamous origin, although adenocarcinoma may occur in the lower esophagus; these arise from esophageal columnar epithelium or from extension of gastric carcinoma into the esophagus. Adenocarcinoma may also develop in esophagus abnormally lined with columnar epithelium. A rare but distinct malignant lesion is carcinosarcoma wherein both squamous carcinoma and sarcomatous elements are present.

Esophageal carcinoma usually presents radiographically as an ulcer, polypoidal mass or annular constriction. Local spread, directly or by local lymphatic chains, may occur vertically or transversely. Vertical spread from the primary lesion via the rich esophageal wall lymphatic bed my occasionally produce secondary implantation sites, resulting in multiple, grossly noncontiguous carcinomas. Lymphatic spread may also occur to paraesophageal, tracheobronchial, supraclavicular and subdiaphragmatic nodes. Direct, vertical submucosal spread is frequent and often extensive. The transverse spread of tumor directly into the mediastinum, trachea, aorta, heart, mediastinal pleura and lungs also is frequent because no serosa surrounds the thoracic esophagus to impede its extension. Tracheal involvement may cause tracheoesophageal fistula and lead to lung abscess. When tumor penetrates the mediastinum, mediastinal abscess may result.

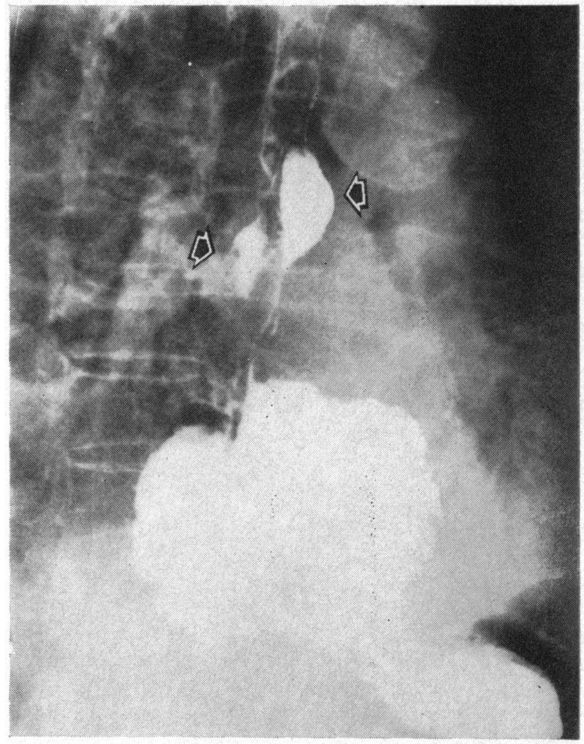

Figure 18.—Peptic ulcers of esophagus. 90-year-old male with a 20-year history of gastroesophageal reflux and recent onset of hematemesis and melena. Barium-air contrast study of the esophagus demonstrated large, penetrating esophageal ulcers (arrows) and a sliding hiatal hernia. At autopsy, the lower esophagus (including the areas of ulceration) was lined by columnar epithelium. The patient died from hemorrhage secondary to erosion of a large atheromatous artery at the base of the smaller ulcer. (Through the courtesy of Dr. F. Dick Berridge, Director, Diagnostic Radiologic Departments, The United Cambridge Hospitals, England.)

When carcinoma originates at or below the carina, 50 percent of patients have subdiaphragmatic node involvement at laparotomy.[20] Mortality is 100 percent when abdominal lymph node metastasis is present. Local extra-esophageal tumor spread is also associated with a grave prognosis. Hematogenous spread to liver, brain, bone, kidneys and adrenal glands may also occur.

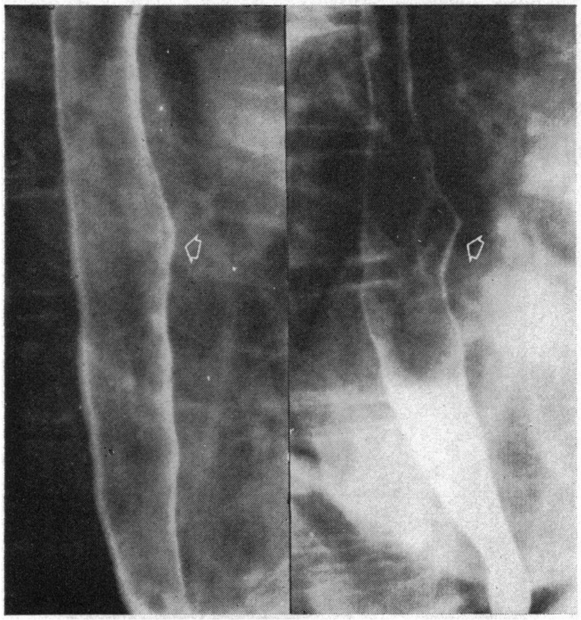

Figure 19. Small esophageal carcinoma. ***Left,*** **slight asymmetry, straightening and rigidity of anterior esophageal wall (arrow) was demonstrated on barium swallow.** ***Right,*** **straight, rigid area (arrow) better defined after the patient was turned slightly.**

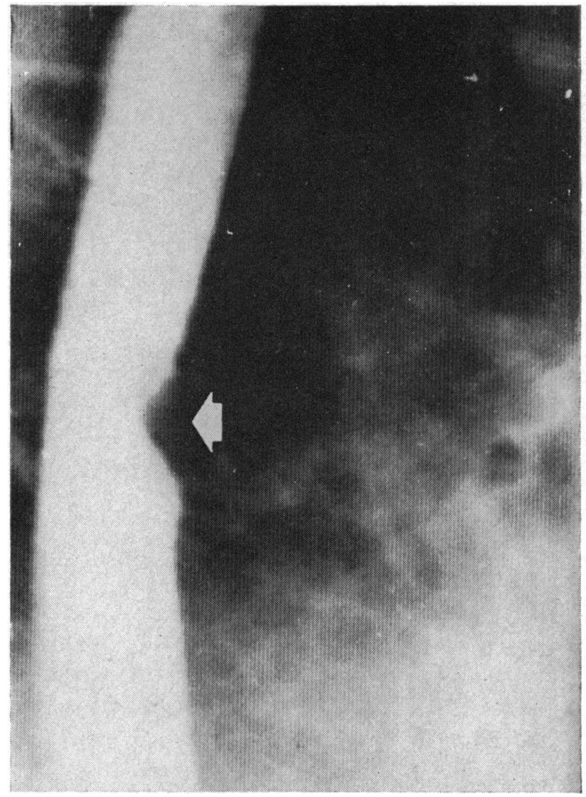

Figure 20.—Small esophageal carcinoma (arrow) presenting as an irregular intraluminal filling defect.

The radiologist's responsibility in esophageal carcinoma includes differentiating malignant tumor from benign tumor or stricture, determining the extent of tumor, and assessing treatment results and complications.

Early esophageal carcinoma rarely causes symptoms. If radiologic examination is done when the lesion is small, it may be difficult to detect. A rigid area (Figure 19) or a small irregular or smooth filling defect should always suggest carcinoma (Figure 20).

Carcinomas present most commonly as annular constricting lesions. The constriction is characterized by a narrow, eccentric, rigid lumen with irregular nodular defects, sharply defined proximal and distal margins which appear "shouldered" and, frequently, tumor mass projecting outside the esophagus (Figure 21). An annular constricting carcinoma may, on occasion, resemble benign stricture from peptic esophagitis.[19] Usually, however, benign stricture has a smooth, concentric lumen, its proximal and distal margins taper gradually and smoothly to join the adjacent normal esophagus and no extrinsic tissue mass is present. Two other criteria may, on occasion, aid differentiation between a benign stricture and malignant constricting lesion.[7] Although both conditions may demonstrate a tortuous esophageal lumen, the rigid lumen of a malignant lesion does not straighten on full inspiration whereas the lumen of benign stricture usually becomes straighter with deep inspiration. Further, a malignant constriction is usually so rigid that cardiac and aortic pulsations displace it whereas the more pliable walls of a benign stricture tend to be indented by such pulsations. When characteristic features exist, differentiation between malignancy and benignancy is easy. At times differentiation is impossible.

Polypoidal esophageal carcinomas are characterized by an irregular intraluminal mass (Figure 20). A carcinomatous ulcer, whether superficial or deep, is usually associated with and superimposed upon tumor mass. The rare carcinosarcoma characteristically is a large, well defined, lobulated, intraluminal mass, but it may present as an annular constricting lesion.

After having diagnosed esophageal carcinoma, the radiologist should attempt to determine the extent of tumor involvement beyond the confines of the esophagus. A number of radiologic techniques aid in determining mediastinal lymph node or tracheal involvement. Enlarged lymph nodes may

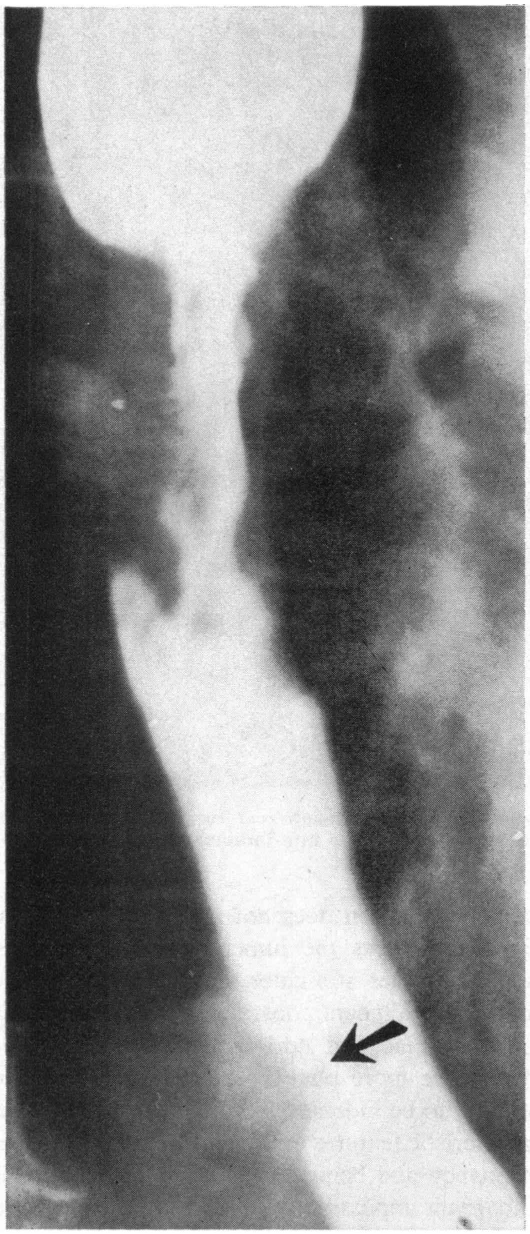

Figure 21.—Annular constricting carcinoma of the esophagus with mediastinal nodal metastastic lesions. An irregular, rigid constriction with "shouldered" edges and an associated soft tissue mass is present just distal to the carina. The extrinsic compression deformity (arrow) below the primary lesion was caused by a mediastinal lymph node, 1.7 cm in diameter, involved by tumor metastasis. (Through courtesy of McGraw-Hill publishers, "Diagnostic Radiology—A Companion to Harrison's Principles of Internal Medicine," P. Ruben Koehler, M.D., editor.)

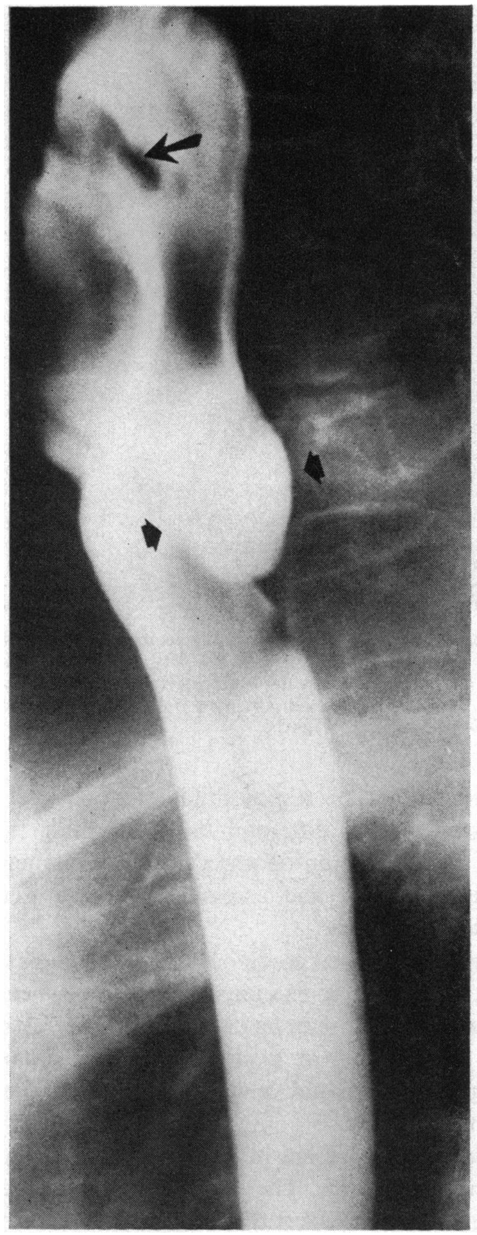

Figure 22.—Zenker's diverticulum. Lateral radiograph demonstrates the barium-filled pharynx (epiglottis identified by large arrow), diverticulum (small arrows) and upper esophagus. The diverticulum has displaced the esophagus forward. Because the diverticulum also lies lateral to the esophagus, it overlaps the barium-filled esophageal lumen. (Through courtesy of McGraw-Hill publishers, "Diagnostic Radiology—A Companion to Harrison's Principles of Internal Medicine," P. Ruben Koehler, M.D., editor.)

cause a smooth, shallow indentation on the barium-filled esophagus distant to the tumor (Figure 21). Lateral chest tomograms and bronchograms may demonstrate tracheal involvement. If tracheo-esophageal fistula occurs, the trachea is always infiltrated with tumor. To determine fistula formation, the patient should swallow thin watery barium while in the prone position.[4] The presence of normal lung fields does not exclude fistula; 75 percent of patients with fistula have no radiologic pulmonary abnormality.[34] Posteroanterior and lateral chest radiographs may demonstrate mediastinal, pulmonary or pleural lesions. Finally, pneumomediastinography may be utilized to delineate tumor extent.[25]

If esophageal carcinoma is treated by radiation therapy, local eradication of tumor cannot be determined by diagnostic radiologic examination. An irregular narrowing usually remains following treatment, and whether this represents radiation esophagitis or residual carcinoma cannot be ascertained with certainty.

Diverticula

Diverticula, although more common in the elderly, may occur at any age. Usually they have little clinical significance. They occur anywhere in the esophagus and frequently are found incidentally during roentgenographic examination. A large diverticulum may present as a posterosuperior mediastinal mass, with or without a fluid level, on a chest roentgenogram. Esophageal diverticula are easily recognized at fluoroscopy. They form saccular luminal outpouchings which vary in size from moment to moment.

A diverticulum may occur in the upper esophageal sphincter (Zenker's diverticulum). It is formed by a mucosal protrusion between the lower transverse and upper oblique cricopharyngeal muscle fibers. About 30 percent of people have a weak area between these muscle layers posteriorly (Killian's dehiscence) through which mucosa may protrude.[35] Because expansion of the diverticulum is limited posteriorly, it deviates laterally and usually to the left; the esophagus is displaced forward and to the right (Figure 22).

Cinefluorography may demonstrate two abnormalities to be associated with Zenker's diverticulum:[2,30,37] (1) inefficient pharyngeal peristalsis and (2) closure of the cricopharyngeal muscle before pharyngeal peristalsis effects pharyngeal emptying.

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